



Challenges and Implications of Dental Caries: A Review

Azuonwu, O., Obire, O., Chukwu, O.O.C., Otimi, E.R

¹Department of Medical Laboratory Sciences, ²Department of Applied and Environmental Biology, Rivers State University of Science and Technology, P.M.B.5080, Port Harcourt, Nigeria.

³Federal College of Veterinary and Medical Laboratory Technology, N.V.R.I, Vom, Nigeria.

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ABSTRACT

Dental Caries is an infectious disease caused by cariogenic microorganisms affecting humans. It is the decay of teeth caused by the action of acid-forming bacteria. A review of public health implication was done to re-emphasize the implications of Dental Caries and the need to build a strong strategy of control and management. The principal causative agents are a group of *Streptococci species* collectively referred to as the *mutans Streptococci* of which *streptococci mutans* are most important agents of human caries. *Streptococcus mutans* increased 6-24 months before the clinical appearance of dental caries. An essential step in caries production appears to be the formation of plaque on the hard, smooth enamel surface and this plaque consist mainly of gelatinous deposits of high-molecular weight glucans in which acid-producing bacteria adhere to the enamel. Dental Caries occurs depending on the genetic, humoral, nutritional, and many other factors. There is a link between caries and weight. Abnormal dietary intake has been linked to the development of obesity and this is common among young people. Obesity is caused by frequent intake of all fermentable carbohydrate foods and drinks whether cooked starches or sugar. Young people are more susceptible to this infectious disease than older ones because children consume more of sucrose than the older people. Worldwide, most children and an estimated 90% of adults have experienced caries with the disease most prevalent in Asia and Latin American countries and least prevalent in African countries. Dental Caries can be controlled by restricting sucrose and other dietary carbohydrates and regular brushing of the teeth, thereby reducing *Streptococcus mutans* and regular check-up with a dental clinic will help for early diagnosis and treatment.

Key words: Plaque, Enamel, Dental Caries, *Streptococcus mutans*.

INTRODUCTION

Dental Caries is a disintegration of the teeth beginning at the surface and progressing inward. The surface enamel, which is entirely non-cellular, is demineralized and this has been attributed to the effect of acid bacterial fermentation. It is caused by the metabolism of the bacteria on plaque attached to the surface of the tooth (1,19). The acid formed by bacterial breakdown the sugar in the diet causing demineralization of the enamel of the tooth and this can lead to tooth loss in children and young adults. The disease majorly affect the crown of the tooth and it initially presents as a white spot in the enamel and on the root as soft areas in the cementum and dentine (2). In recent time, the prevalence of Dental Caries in the most western countries has steadily declined. By contrast in some developing Countries such as Nigeria, Zambia, Indonesia, Sudan and Thailand have indicated a marked increase in Dental Caries because of the high intake of sucrose (31).

DESCRIPTION OF DENTAL CARIES

Teeth are composed of thin layer (1-2mm) of dental enamel which forms the hard protective coating over the tooth (2,8). This consists mainly of calcium, phosphate, and other ions in a structure known as "hydroxyapatite". Dental enamel is porous and is susceptible to acid dissolution during the process of remineralization. Salivary component which can be critical in controlling Dental Caries since structure can neutralize acids. Saliva has a vital role in the demineralization and re-mineralization of the enamel and determines whether Caries occurs (17,24). Saliva protects the teeth against acid dissolution and also acts quickly to clear away food debris from the mouth and to buffer the organic acids that are produced by the bacteria.

SCIENTIFIC CLASSIFICATION OF DENTAL CARIES

Kingdom : Bacteria
Phylum : Firmicutes
Class : Cocci
Order : Lactobacillales
Family : Streptococcaceae
Genus : Streptococcus
Specie : Mutans

*Corresponding author.

Azuonwu, Obioma.
Department of Medical Laboratory Science,
Rivers State University of Science and Technology,
Nkpolu, P.M.B 5080, Port Harcourt, Rivers State, Nigeria.
Tel: +234803519688.
Email: azuonwuobi@yahoo.com

Binomial : *Streptococcus mutans*

PUBLIC HEALTH IMPLICATIONS OF DENTAL CARIES

There is a link between caries and weight. Abnormal dietary intake has been linked to the development of obesity and this is common among young peoples. Obesity is caused by frequent intake of all fermentable carbohydrate foods and drinks whether cooked starches or sugar (6,10,16,30). Coloration of the teeth is another implication of Dental Caries and the person will not be able to open his or her teeth in the public due to the color of the teeth (20). Also, another implication of Dental Caries is mouth odour (9). Mouth odour is caused through the ability of a food to be retained in the mouth which will alter its potential carcinogenicity (26,31). Starchy foods are retained longer in the oral cavity than some of the fast clearance sugary foods (12). Someone with mouth odour will not have confidence enough to talk in the public. This will affect the relationship of the person with people (6).

CAUSES OF DENTAL CARIES

The first report of the involvement of Streptococci in the aetiology of Dental Caries was in 1924 by Clarke. He isolated Streptococci with distinctive characteristics and named them *Streptococcus mutans*. Bacteria previously referred to as *Streptococcus mutans* are now subdivided into seven distinct species (15). These species are often collectively referred to as the mutans streptococci (MS) because they have a number of common properties relevant to Caries inducing ability. These species are found in the oral cavity and it is more convenient to refer to them as a cluster rather than as individual species (6). The involvement of specific bacteria in Caries development is referred to as the 'specific plaque hypothesis'. *Streptococcus mutans* increased 6-24 months before the clinical appearance of Dental Caries. The most active Caries bacteria among many types of bacteria are: *Streptococcus mutans*, *Lactobacillus spp.*, *Veillonella spp.*, and *Actinomyces spp.* (20). Mutans Streptococci can ferment various sugars to produce lactic acid. Lactate dehydrogenase (LDH) enzymes convert propionate to lactate, but when Carbohydrate is limited these bacteria produce formate, acetate and ethanol. Prevalence of Dental Caries is high in immune-compromised patients because their immune system is not strong enough to attack any invading organism (19).

CARIOGENIC ATTRIBUTES OF MUTANS STREPTOCOCCI

Mutans streptococci possess certain properties that enable it to predominant in Dental Caries (8). These include extracellular polysaccharide synthesis, acidogenicity (acid production), acidity (ability to survive in an acidic envi-

ronment), intracellular polysaccharide synthesis and endodextrans production (13).

SYMPTOMS

Dental Caries is usually far advanced before any symptoms develop (1). The severe, throbbing pain of a toothache is often the first symptom. Sometimes there is noticeable discoloration, roughness, or defeat, and a tooth can break during chewing of strong foods (6).

LABORATORY DIAGNOSIS

1.The appropriate specimen for the diagnosis of Dental caries is obtained by collecting abscesses or pulp material from the gum which is carefully aspirated by needle and syringe, after decontamination of the surface with providone iodine(3).

2.A gram stain is first performed which should be typical gram positive cocci in chains.

3.Culture the organisms on a blood agar which is a selective medium and incubate under CO₂-enriched atmosphere at 37°C for 48-72 hours, the agar plates were inspected for growth of mutans Streptococci (4,11). Blood agar will support the growth of wide variety of bacteria. Haemolysis (alpha and beta) can often be used to aid in the identification of some organisms that will grow on this medium. *Streptococcus mutans* are facultative anaerobic bacteria that are spherical or ovoid with less than 2 micrometer in diameter (13,14). Other two selective media that are widely used for isolating Canes-related streptococci are based on Mitissalivacus agar and trypticase yeast Columbia blood (TYCB) agar. This suppresses the growth of most species but allow *Streptococcus mutans* to grow. Streptococcus mutaris is catalase negative (-) and is sensitive to penicillin, and also sensitive to metronidazole (23,25).

PATHOGENESIS

The first step in the formation of a cariogenic plaque, meaning a plaque that causes tooth decay, is the adherence of oral Streptococci to specific reception on the tooth pellicle. The pellicle is a thin film of proteinaceous material adsorbed on the tooth from the saliva. Sucrose is split by the enzymes to the monosaccharide's glucose and fructose. The glucose is polymenized; yielding glucans bind the organism together and to the tooth to form a bio-firm, making a plaque impenetrable to saliva (17). When sugar enters the mouth, the pH of cariogenic plaques drops from its normal value of about 7 to below 5 within minutes. The duration of this acidic state depends on how long the teeth have been exposed to sugars and on the concentration of the sugars. After food leaves the mouth, the pH of the plaque rises slowly to neutrality

PICTORIAL REPRESENTATION OF DENTAL CARIES



Fig.1



Fig.2



Fig.3

(35). The delay in return of the pH to neutrality is due to the ability of *Streptococcus mutans* to store a portion of its food as an intracellular, starch like polysacchandes that is later metabolized with the production of acid. Cariogenic plaque thus acts as a tiny, acid-soaked sponge closely applied to the tooth. Pathologically, low salivary flow leads to Dental Caries(7).

EPIDEMIOLOGY

The Distribution of Dental Caries varies in different parts of the world and within the same country or region. Worldwide, most children and an estimated 90% of adults have experienced Caries with the disease most prevalent in Asia and Latin American Countries and least prevalent in African countries (33). In the United States, Dental Caries is the most common chronic childhood disease, being at least 5 times more common than asthma. It is the primary pathological cause of tooth loss in children. About 29% to 59% of adults over the age of 50 experience Caries (18,19).

Nonetheless, countries that have experienced an overall decrease in cases of tooth decay continue to have a disparity in the distribution of the disease (32). Among children in the United States and Europe, twenty percent of the population endures 60- 80% of cases of Dental Caries. Australia, Nepal and Sweden have a low incidence of cases and more numerous in Costa Rica and Slovakia. It was also noted that females experience more decay as compared to males (26).

PREVENTION

The most important method for preventing Dental Caries is restricting sucrose and other refined dietary carbohydrates, thereby reducing *Streptococcus mutans* colonization of teeth and acid production by cariogenic plaques. Dental Caries can be reduced by cariogenic plaques. Dental Caries can be reduced by 90% if sucrose-containing sweets are eliminated from the diet (5). It is not however simply the quantity of sugar in the diet that is important. Interestingly, chewing paraffin or sorbitol-sweetened gum reduces Dental Caries, probably because it increases the flow of saliva. Trace amounts of fluoride are required for teeth to resist the acid of cariogenic plaques (26).

Fluoride makes tooth enamel harder and more resistance to dissolving in acid (22). Mechanical removal of plaque by tooth brushing and use of dental floss is another important preventive measure, reducing the incidence of dental Caries by about 50%(16). Caries commonly develops in pits and fissures can be prevented by using a sealant (a kind of epoxy that seals the fissures). The sealant kills the bacteria in plaque and prevents bacterial re-colonization (3,8). Regular visit to the dentist for regular checkups and professional plaque removal prevents Dental Caries (20,34).

TREATMENT

Treatment of Dental Caries requires drilling out the cavity, filling the defect with amalgam an alloy of mercury and some other metal of other material, and restoring the contour of the teeth(8). Dental Caries can also be treated by application of fluoride, application of chiohexedine, using sealant antimicrobials, salivary enhancer and patient education (16,23). Chiorhexedine is appropriately used for primary prevention (36).

CONCLUSION

Streptococcus mutans is the principal aetiological agents of dental Caries which colonize the teeth soon after they erupt. Cariogenic features of these bacteria include synthesis of water- soluble glucans, lactic acid production, ability to survive at a low pH, intracellular polysaccharide synthesis and the production of a dextran-hydrolyzing enzyme (endodextranase). Fluoride is still the best available anti-caries chemical agent, its anti-caries action is attributable to increasing the resistance of the tooth to acid demineralization, stimulation of remineralization and inhibition of mutans streptococci carbohydrate metabolism. Caries is endemic and potentially both preventable and curable.

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REFERENCES

1. Anderson P.F, (2001): Search Strategies for Independent Reviewers. *Journal Dental Education* 65:1169-1179.
2. Ash and Nelson, (2003): *Wheeler's Dental Anatomy, Physiology, And Occlusion* 8th Edition, Saunders.
3. Bader J, (2001): *Diagnosis and Management of Dental Caries*. Agency for Health care Research and Quality.
4. Banting D.W. (2001): The Diagnosis of Root Caries: *Journal Dental Education* 65:991-996.
5. Batchelor P and Sheiham A (2002): The limitations of a "high risk" approach for the prevention of Dental Caries. *Community Dentistry Oral Epidemiology* 30:302-12.
6. Burt B. and Pai S (2001): Sugar consumption and canes risk: a Systemic Review. *Journal Dental Education* 65:1017-23.
7. Dye B.A, Shenkin J.D, Ogden C.L, Marshall T.A, Levy S.M and Kanellis M.J. (2004): The Relationship between Healthful Eating practices and Dental Caries in Children aged 2-5 years in United States; 35:55-66.
8. Fejerskov and Ole, (2008). *Dental Caries: The Disease and its Clinical Management*. Oxford, Blackwell Munksgaard.
9. Gidding S.S, Dennison B.A, Birch L.L, Daniel S.R, Gilman M.W and Lichtenstein A.H. (2005): American Heart Association Dietary Recommendations for Children and Adolescents.
10. Hilgers K.K, Kinane D.E and Schetz J.P. (2006): Association Between Childhood and Obesity and Smooth Surface Caries in Posterior Teeth: a preliminary Study. *Pediatric Dentistry*; 28:23-8.
11. Horowitz A.M, (2001): A Report on National Institute of Health Consensus Development Conference on Diagnosis and Management of Dental Caries throughout life. *Journal of Dental Research* Huang E.A, Becerra k, Walker E, and Hovell M.F. (2006): A Childhood Overweight and Orthodontists: Result of Survey. *Public Health Dentistry* 66:292-4.
13. Kidd E.A, (2001): Diagnosis of Secondary Caries. *Journal of a Survey: Public Health Dentistry* 66:292-4.
14. Kidd E.A, (2001): Diagnosis of Secondary Caries. *Journal Dental Education* 65:997-1000.
15. Kidd E.A, (2005): *Essentials of Dental Caries*. Oxford University Press. ISBN 0198529783.
16. Moynihan P. and Petersen P.E, (2004): Diet, nutrition and the Prevention of Dental Disease. *Public Health Nutrition* 7:20 1-26.
17. Neville B.W, Douglas Damm, Carl Allen and Jerry Bouquot, (2002). *Oral and Maxillofacial Pathology*, 2nd Edition. ISBN 0-72 16-900-3.
18. Peterson D.E, (2004): Improvement of oral health in Africa in The 21st century- the role of the world health Organizations. *Global Oral Health Programme. Developing Dentistry*; 5-11.
19. Petersen P.E, (2003): The World Oral Health Report the Improvement of Oral Health in the 21st Century- the approach of the WHO Global Oral Health Programme. *Community Dentistry Oral Epidemiology* 31:3-24.
20. Petersen P.E, Kjoller M, Christensen LB and Krstrup, (2004): Changing dentate status of adults. Use of Dental Health Services and achievement of national Dental health goals. *Journal of Public Health* 64 127-35.
21. Petersen P.E, Hoerup N, Poomviset N, Wantanapa and Prommajan J, (2001): Oral health status and Oral Health behavior of urban and rural school Children in southern Thailand. *International Dentistry Journal* 5 1:95-102.
22. Petersen P.E, Nyandindi U, Kikwilo E, Mabelya L, Lembariti BS, Poulson V.J, (2002): Oral health status and Oral Health behavior of urban and rural school Children, teachers, and adults in Tanzania.
23. Pitts N.B, (2001). *Clinical Diagnosis of Dental Caries: A European Perspective*. *Journal Dental Education* 65:972-978.
24. Rogers and Anthony H, (2008): *Molecular Oral Microbiology*. Caister Academic press.
25. Shugars D, Bader J and Bonito A, (2002): *Emerging Technologies for Diagnosis of Dental Caries: the Road so Far*.
26. Sogi G and Bhasker D.J, (2001): Dental Caries and Oral hygiene Status of 13-14 years old schools children of Davangen. *Journal of Indian Society of Pedodontic and preventive Dentistry*: 113-117.
27. Sonis, Stephen I, (2003): *Dental Secrets: Questions and Answers Reveal the Secrets to the principles and Practice of Dentistry*, 3rd edition. ISBN 1- 56053-57 3-3.
28. Sundby A and Petersen PE, (2003): Oral health status in Relation to ethnicity of children in the municipality Of Copenhagen, Denmark. *International Journal Pediatric Dentistry* 13:150-7.
29. Tencate J.M, Lagerweil M.D, Wefel J.S, Angmar- Mansson B, Hall A.F and Ferreira-zandona A.G, (2000): In Early Detection of Dental caries. *Proceedings of the 4th Annual Indiana Conference*; Indiana University Bloomington pg 231-259.
30. Vann W.F, Bouwens T.J, Braithwaite A.S and lee J.Y, (2005): The Childhood Obesity Epidemic: A Role for Pediatric Dentists; 27:271-6.
31. Wilier Shausen B, Hass G, Kerummenauer F, Hohenfelirer K, (2004): Relationship between Weight and Caries Frequency in German Elementary School Children 9:400-4.
32. Woodmansay K.F, (2005): The prevalence of Dental Canes among international students at United States Universities. *Journal of Contemporary Dental Practice*. 6:124-135.
33. World Health Organization, (2003): *Continuous Improvement Of oral health in the century — Global Oral Health Programme: World Oral Health*.
34. World Health Organization, (2002): *The World Health Report: Reducing risks, promoting health life*.
35. World Health Organization, (2003): *Oral Health Surveys Basic Methods*. 4th edition Geneva. World Health Organization.
36. Zhu L, Petersen PE, Wang HY, Bian JY and Zhang BX, (2003): Oral health knowledge, attitudes and behavior Of Children and adolescent in china. *International Dentistry Journal* 53:289-98.

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